SO NEPHRON, (JAN 1992) Vol. 60, No. 1, pp. 42-48.
ISSN: 0028-2766.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 33
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We have shown that the inhibition of prostaglandin (PG) synthesis in man decreases the fractional clearance of urea (FC(urea)). To understand the mechanism(s) by which PG affect the renal handling of urea, 6 normal volunteers were randomly studied in maximal antidiuresis (by water deprivation and by **administering** 1-desamino-8-D-**arginine** vasopressin) before and during PGE, infusion, in two separate occasions: (A) after 7 days of normal protein (1 g/kg b.w./day) and water intake (10 ml/kg b.w./day), and (B) after 7 days of low protein intake (0.5 g/kg b.w./day) and high water intake (80 ml/kg b.w./day) to lower the corticomedullary osmotic gradient. During infusion of PGE1 at rates of 0.01, 0,05 and 0.1-mu-g/min/kg, randomly administered, the urinary fluid losses were replaced by infusing equal volumes of hypotonic NaCl (80 mmol/l). To evaluate the time effects of this protocol, control studies were performed in an other 8 subjects receiving vehicle infusion without PGE1. In study A, FC(urea) rose by 23% ($p < 0.01$) at the lowest PGE1 infusion rate (0.01-mu-g/min/kg), in the absence of any simultaneous change in water and salt output, Uosm, PAH and inulin clearance. Higher PGE, infusion rates (0.05 and 0.1-mu-g/min/kg) were associated with a progressive increase of FC(urea) (50%, $p < 0.001$ and 91%, $p < 0.001$, respectively), fractional clearance of water and salt output, inulin and PAH clearance and reduced Uosm from 1,005 (22 SEM; basal value) to 772 (38 SEM; minimum value) mosm/kg ($p < 0.001$). In study B, the basal value of Uosm was 762 (22 SEM) mosm/kg, markedly lower than the basal value of study A ($p < 0.01$); in this condition, the increasing infusion rates of PGE1 caused the same changes of FC(urea) and the other parameters as in study A. FC(urea) was directly related to dose infusion of PGE1 both in study A and B ($p < 0.001$). The slopes of these two linear regression analyses did not statistically differ. Finally, both FC(urea) and fractional clearance of water did not show significant changes among the several periods of the control studies. We conclude that in human subjects, the inhibition of urea tubular reabsorption, observed during PGE1 infusion, is: (1) not associated with change in tubular handling of salt and water at the lowest infusion of PGE1; (2) not mediated by passive hydrosmotic forces or by antagonism with ADH; (3) dependent on the dose of exogenous PGE1.

L12 ANSWER 1977 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:637983 SCISEARCH
GA The Genuine Article (R) Number: GP919
TI EFFECTS OF NEONATAL ADMINISTRATION OF VASOPRESSIN ON CARDIAC AND BEHAVIORAL-RESPONSES TO EMOTIONAL-STRESS IN ADULT MALE-RATS
AU BUWALDA B (Reprint); NYAKAS C; KOOLHAAS J M; BOHUS B
CS UNIV GRONINGEN, CTR BEHAV COGNIT & NEUROSCI, DEPT ANIM PHYSIOL, POB 14, 9750 AA HAREN, NETHERLANDS (Reprint)
CYA NETHERLANDS
SO PHYSIOLOGY & BEHAVIOR, (1991) Vol. 50, No. 5, pp. 929-932.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 28
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB **Arginine**-8-vasopressin (AVP) was **administered** subcutaneously on postnatal days 3-7 in a high (10-mu-g/100 g b. wt.) or a low dose (1-mu-g/100 g b. wt.) to male Wistar rats. Control pups were untreated or saline injected. Behavioral observations in a complex maze after maturation indicated that neonatal administration of AVP increases exploratory behavior in this novel environment in a dose-dependent way.

Cardiac monitoring during the conditioned emotional stress of fear of inescapable electric footshock showed that only the high dose of AVP attenuates the bradycardiac stress response. The analysis of cardiac responses also suggested an adult hyposensitivity to AVP in rats treated neonatally with AVP. In addition, the low dose of neonatal AVP was impairing the retention of a passive avoidance behavior. The data indicate that the neonatal administration of AVP exerts long-term effects upon the behavioral adaptation to novelty and memory processes related to emotional stress. That neonatal AVP is less effective in influencing adult vagally mediated cardiac stress responses suggests differences in the developmental sensitivity ("critical periods") of the central vasopressinergic systems involved in the regulation of behavior and autonomic functioning.

L12 ANSWER 1978 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:624794 SCISEARCH
GA The Genuine Article (R) Number: GP397
TI EFFECTS OF THE NITRIC-OXIDE SYNTHASE INHIBITOR NG-NITRO-L-ARGININE ON THE ERECTILE RESPONSE TO CAVERNOUS NERVE-STIMULATION IN THE RABBIT
AU HOLMQUIST F (Reprint); STIEF C G; JONAS U; ANDERSSON K E
CS UNIV LUND HOSP, DEPT CLIN PHARMACOL, S-22185 LUND, SWEDEN (Reprint); HANOVER UNIV MED, DEPT UROL, HANNOVER, GERMANY
CYA SWEDEN; GERMANY
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1991) Vol. 143, No. 3, pp. 299-304.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 17
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Using a rabbit model, the involvement of the L-arginine/nitric oxide pathway in penile erection was investigated. The mean basal intracavernous pressure was 21 cm H2O. Cavernous nerve stimulation (4-8 V, 20-30 Hz) increased the pressure to approximately 130 cm H2O. This response was highly reproducible and usually associated with full penile erection. The pressure increase could be quantified in terms of: (1) the slope of the initial, ascending part of the pressure increase; (2) DELTA-P, which was defined as the maximal pressure obtained by the stimulation minus the basal pressure before the stimulation; (3) T90, which was defined as the time to reach 90 per cent of DELTA-P. Intrapenile administration of the L-arginine/nitric oxide synthesis inhibitor N(G)-nitro-L-arginine had no effect on systemic arterial blood pressure. However, N(G)-nitro-L-**arginine** (0.22 and 2.19 mg), **administered** via the same route, abolished the erectile response induced by cavernous nerve stimulation; T90 increased and slope and DELTA-P decreased significantly. N(G)-nitro-D-arginine (2.19), on the other hand, had no inhibitory effect. L-arginine (21.07 mg), given either directly or after N(G)-nitro-L-arginine had no consistent effect on the functional response to cavernous nerve stimulation.

The results suggest that pharmacologically induced effects on intracavernous pressure in the rabbit can be described quantitatively, and that this model may be useful to study the mechanisms controlling penile erection in vivo. The pronounced inhibitory action of N(G)-nitro-L-arginine demonstrates the important role of the arginine/nitric oxide pathway in mediating relaxation of penile smooth muscles necessary for erection.

L12 ANSWER 1979 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:600420 SCISEARCH
GA The Genuine Article (R) Number: GM439
TI EFFECT OF L-ARGININE AND AN ARGININE-CONTAINING PENTAPEPTIDE ON CANINE FEMORAL ARTERIAL BLOOD-FLOW
AU SALDEEN K (Reprint); NICHOLS W W; NICOLINI F; MEHTA J
CS UNIV FLORIDA, COLL MED, DEPT MED, GAINESVILLE, FL, 32611; LINKOPING UNIV, FAC HLTH SCI, DEPT PHARMACOL, S-58183 LINKOPING, SWEDEN; UNIV UPPSALA,

DEPT FORENS MED, S-75105 UPPSALA, SWEDEN
CYA USA; SWEDEN
SO UPSALA JOURNAL OF MEDICAL SCIENCES, (1991) Vol. 96, No. 2, pp. 113-118.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 8
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The amino acid L-arginine is a precursor of endothelium derived relaxing factor (EDRF). The pentapeptide 6A (Ala-Arg-Pro-Ala-Lys) released by plasmin degradadation of fibrinogen also contains arginine and relaxes vascular smooth muscle by releasing EDRF (nitric oxide). To determine and compare the effects of L-arginine, peptide 6A and a combination of L-arginine and peptide 6A on femoral artery blood flow and vascular resistance, anesthetized mongrel dog were **administered** saline, L-**arginine**, D-**arginine**, peptide 6A and L-arginine + peptide 6A in a random order.

L-arginine and peptide 6A both induced an immediate dose-dependent short-lasting increase in femoral blood flow and a decrease in vascular resistance. Peptide 6A exerted a much greater ($P < 0.01$) vasodilatory effect than did L-arginine at the same molar concentration suggesting that properties besides the arginine content are important in the effect of the pentapeptide. D-arginine had much less effect than L-arginine, indicating that the effect of L-arginine may be related to its utilization for synthesis of EDRF. When the peptide 6A was given soon after L-arginine, its effect on blood flow was not greater than that of L-arginine alone suggesting that L-arginine in a large amount makes guanylate cyclase less available for the more active peptide.

L12 ANSWER 1980 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:500043 SCISEARCH
GA The Genuine Article (R) Number: GD283
TI REGIONAL CORONARY HEMODYNAMIC-EFFECTS OF 2 INHIBITORS OF NITRIC-OXIDE SYNTHESIS IN ANESTHETIZED, OPEN-CHEST DOGS
AU RICHARD V; BERDEAUX A; LAROCHELLE C D; GIUDICELLI J F (Reprint)
CS FAC MED PARIS SUD, PHARMACOL LAB, 63 RUE GABRIEL PERI, F-94276 LE KREMLIN BICETR, FRANCE
CYA FRANCE
SO BRITISH JOURNAL OF PHARMACOLOGY, (1991) Vol. 104, No. 1, pp. 59-64.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 27
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB 1 The role of endothelial nitric oxide synthesis from L-arginine in the regulation of coronary vascular tone and myocardial tissue perfusion was evaluated in anaesthetized, open-chest dogs. Coronary blood flow was measured with an electromagnetic flow probe placed around the left circumflex coronary artery. Coronary vascular resistance was calculated from mean arterial blood pressure and mean coronary blood flow, whereas regional myocardial tissue flow was determined by use of the radioactive microspheres technique.

2 N(G)-monomethyl L-arginine (L-NMMA) and N(G)-nitro-L-**arginine** methyl ester (L-NAME), **administered** directly into the left circumflex artery, induced a small increase in arterial blood pressure and an increase in coronary vascular resistance. However, myocardial tissue perfusion, assessed by the microspheres technique (whether subendocardial, subepicardial, or transmural), was unaffected by L-NMMA or L-NAME.

3 Acetylcholine, administered intracoronarily, induced an increase in left circumflex coronary blood flow and a decrease in coronary vascular resistance, without affecting systemic haemodynamics. This coronary vasodilator effect of acetylcholine was markedly inhibited by L-NMMA and L-NAME, the latter being a more potent antagonist than the former.

4 These results indicate that the endothelial L-arginine pathway is largely responsible for the coronary vasodilator effect of acetylcholine. However, although basal release of nitric oxide from L-arginine apparently contributes to the regulation of resting coronary vascular tone, blockade of this pathway does not affect myocardial tissue perfusion, possibly because of compensatory mechanisms occurring at the level of small arterioles and/or capillaries.

L12 ANSWER 1981 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:441362 SCISEARCH
GA The Genuine Article (R) Number: FZ154
TI CENTRALLY **ADMINISTERED** GALANIN INHIBITS OSMOTICALLY STIMULATED **ARGININE** VASOPRESSIN RELEASE IN CONSCIOUS RATS
AU KONDO K (Reprint); MURASE T; OTAKE K; ITO M; OISO Y
CS NAGOYA UNIV, SCH MED, DEPT INTERNAL MED 1, 65 TSURUMAI CHO, SHOWA KU, NAGOYA, AICHI 466, JAPAN (Reprint)
CYA JAPAN
SO NEUROSCIENCE LETTERS, (1991) Vol. 128, No. 2, pp. 245-248.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 22
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The effect of centrally **administered** galanin on **arginine** vasopressin (AVP) release was investigated in conscious rats. Intracerebroventricular injection of porcine galanin suppressed hypertonic saline-induced increase in plasma AVP in a dose-dependent manner (12.5-100 pmol/rat) at 10 min after the injection. Pretreatment with subcutaneous injection of naloxone (1 mg/100 g b.wt.) partially blocked the galanin-induced effect on plasma AVP. These results suggest that central galanin inhibits osmotically stimulated AVP release and endogenous opioids are, at least in part, involved in the mechanism.

L12 ANSWER 1982 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:410253 SCISEARCH
GA The Genuine Article (R) Number: FW523
TI SOCIAL-STATUS IN PAIRS OF MALE SQUIRREL-MONKEYS DETERMINES THE BEHAVIORAL-RESPONSE TO CENTRAL OXYTOCIN ADMINISTRATION
AU WINSLOW J T (Reprint); INSEL T R
CS NIMH, NIHAC, CLIN SCI LAB, POB 289, POOLESVILLE, MD, 20837 (Reprint)
CYA USA
SO JOURNAL OF NEUROSCIENCE, (1991) Vol. 11, No. 7, pp. 2032-2038.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 47
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Oxytocin, when administered centrally, has been associated with the modulation of various social initiatives including maternal and sexual behaviors. The nature of these effects depends on gonadal hormone status. In the present experiments, we investigated the effects of centrally administered oxytocin on the behavior of pair-housed male squirrel monkeys during interactions with a familiar female monkey. Pairs of male squirrel monkeys established reliable and persistent dominance relationships with dominant males showing increased sexual and aggressive behavior as well as higher plasma concentrations of testosterone. Oxytocin (0.1, 1.0-mu-g) increased the sexual and aggressive behavior of dominant monkeys without affecting these measures in the sub-ordinate monkeys. In contrast to these effects in the dominant monkeys, oxytocin increased associative and marking behaviors only in subordinate monkeys. Central administration of the oxytocin receptor antagonist d(CH2)5[Tyr(Me)2, Thr4,Tyr-NH2(9)] OVT (OTA; 0.05-mu-g) had no intrinsic effect on behavior but blocked the effects of exogenous oxytocin. To investigate further the specificity of oxytocin's effects on social behavior, we **administered** the

structurally related peptide **arginine** vasopressin under identical conditions. Vasopressin (0.5, 5.0-mu-g) decreased social behaviors and increased motor activity in both dominant and subordinate monkeys. Previous studies in rodents have demonstrated that oxytocin receptors are induced by gonadal steroids in a regionally specific fashion. The status-related behavioral effects of oxytocin in the squirrel monkey may reflect differences in brain oxytocin receptor density associated with the higher concentrations of testosterone in the dominant animal. Alternatively, the status-related effects may depend on the conditioned behavioral differences associated with social organization.

L12 ANSWER 1983 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:351979 SCISEARCH
GA The Genuine Article (R) Number: FR212
TI DIETARY ARGININE FAILS TO PROTECT AGAINST CEREBROVASCULAR DAMAGE IN STROKE-PRONE HYPERTENSIVE RATS
AU STIER C T (Reprint); SIM G J; LEVINE S
CS NEW YORK MED COLL, DEPT PHARMACOL, BASIC SCI BLDG, VALHALLA, NY, 10595 (Reprint); NEW YORK MED COLL, DEPT PATHOL, VALHALLA, NY, 10595
CYA USA
SO BRAIN RESEARCH, (1991) Vol. 549, No. 2, pp. 354-356.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 11
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Stroke-prone spontaneously hypertensive rats (SHRSP) develop severe hypertension and cerebrovascular lesions. We investigated the influence of dietary supplementation with L-arginine, an amino acid precursor of endothelium-derived nitric oxide, on blood pressure and stroke in these rats. L-**Arginine**, **administered** in the saline drinking solution at 2 or 6 g/l starting at 8.7 weeks of age, was without effect on blood pressure, cerebrovascular lesions, or longevity despite continuous treatment through 14 weeks of age. These findings do not support a beneficial influence of dietary arginine in the cerebrovascular pathology of SHRSP.

L12 ANSWER 1984 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:350358 SCISEARCH
GA The Genuine Article (R) Number: FR351
TI EVIDENCE THAT L-ARGININE POSSESSES PROCONVULSANT EFFECTS MEDIATED THROUGH NITRIC-OXIDE
AU MOLLACE V; BAGETTA G; NISTICO G (Reprint)
CS UNIV ROME TOR VERGATA, DEPT BIOL, CHAIR PHARMACOL, VIA ORAZIO RAIMONDO, I-00173 ROME, ITALY
CYA ITALY
SO NEUROREPORT, (1991) Vol. 2, No. 5, pp. 269-272.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 22
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB INCREASING evidence suggests a neurotransmitter role for NO in the mammalian CNS. We have now studied the behavioural and electrocortical (ECoG) profile of rats injected into the lateral cerebral ventricle (ICV) with L-arginine (L-arg), the endogenous donor of the guanidino group from which NO physiologically originates. Rats treated with L-arg (up to 300-mu-g) showed behavioural stimulation, ECoG desynchronization with occasional isolated high voltage spikes but not motor seizures. In rats receiving a subconvulsive dose (0.5-mu-g) of N-methyl-D-aspartic acid, (NMDA; ICV) the microinjection of L-arg (300-mu-g; 1 min before) resulted in behavioural and ECoG seizures. The latter effects were prevented by co-**administering** L-arg with N-nitro-L-**arginine** (L-NAME), an inhibitor of NO synthesis. In conclusion, L-arg possesses

proconvulsant effects probably mediated by an increase in NO synthesis.

L12 ANSWER 1985 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:253480 SCISEARCH
GA The Genuine Article (R) Number: FH481
TI STUDIES ON THE PRECURSOR OF METHYLGUANIDINE IN RATS WITH RENAL-FAILURE
AU YOKOZAWA T (Reprint); FUJITSUKA N; OURA H
CS TOYAMA MED & PHARMACEUT UNIV, DEPT APPL BIOCHEM, WAKAN YAKU RES INST, SUGITANI, TOYAMA 93001, JAPAN (Reprint)
CYA JAPAN
SO NEPHRON, (1991) Vol. 58, No. 1, pp. 90-94.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 27
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Each of creatinine (Cr), guanidinoacetic acid (GAA) and **arginine** (Arg) was **administered** intraperitoneally to rats with renal failure, and the levels of methylguanidine (MG) in the serum, liver, kidney, muscle and urine were determined at certain intervals. The levels of MG in the serum, liver, kidney, muscle and urine after Cr administration increased markedly with time. The amount of total MG at 24 h was estimated to be 114-mu-g/100 g body weight, which accounted for 0.46% of the Cr dose. In contrast, changes in MG levels after administration of GAA or Arg were only slight in comparison with those after Cr administration. Thus, MG was proved to be produced mainly from Cr.

L12 ANSWER 1986 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:176046 SCISEARCH
GA The Genuine Article (R) Number: FC708
TI EFFECT OF EXOGENOUS ARGININE VASOPRESSIN ON ADRENOCORTICOTROPIN AND CORTISOL RELEASE IN MYOTONIC-DYSTROPHY PATIENTS - DELAYED-RESPONSES OF NORMAL MAGNITUDE
AU GRICE J E; JACKSON J; HEWETT M; PENFOLD P J; JACKSON R V (Reprint)
CS UNIV QUEENSLAND, GREENSLOPES HOSP, DEPT MED, NEUROENDOCRINE RES UNIT, BRISBANE, QLD 4120, AUSTRALIA
CYA AUSTRALIA
SO JOURNAL OF NEUROENDOCRINOLOGY, (1991) Vol. 3, No. 1, pp. 65-68.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 24
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We **administered** intramuscular **arginine** vasopressin (AVP) to ten normal controls and eight myotonic dystrophy patients. By measuring plasma AVP levels in five of the myotonics and all the normals, we showed that absorption and distribution of AVP was not delayed or significantly reduced in myotonics. The magnitude of the mean plasma adrenocorticotropin (ACTH) response to AVP in the myotonics was not different from that of normals, although it was significantly delayed (mean peak time, 37.5 +/- 4.9 versus 17.0 +/- 3.2 min). We propose that this delay was caused by a significantly reduced ACTH secretion rate in myotonics, because the maximum rate of detection of ACTH in plasma is reduced in myotonics (0.6 +/- 0.2 versus 1.7 +/- 0.5 pmol/L/min), whose corticotropes, while having the same capacity to respond to the AVP stimulus, are slower to attain that capacity. The mean integrated cortisol response (AUC) was significantly smaller for myotonics (8072 +/- 2017 versus 13049 +/- 1630 nmol.min/L). This may be due to the slower rate of ACTH delivery to the adrenal in myotonics. The timing of the adrenal response does not appear to be impaired in myotonic dystrophy, with the cortisol peak following the ACTH peak by approximately 15 min in both groups. The normal magnitude ACTH response to AVP in myotonics is in contrast to that seen to ACTH secretagogues acting via

corticotropin-releasing hormone-initiated pathways, where a rapid hypersecretion is seen. We propose a mechanism of defective calcium transport to account for these observations.

L12 ANSWER 1987 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:54404 SCISEARCH
GA The Genuine Article (R) Number: ET513
TI NITRIC-OXIDE REQUIREMENT FOR VASOMOTOR NERVE-INDUCED VASODILATATION AND MODULATION OF RESTING BLOOD-FLOW IN MUSCLE MICROCIRCULATION
AU PERSSON M G (Reprint); WIKLUND N P; GUSTAFSSON L E
CS KAROLINSKA INST, KAROLINSKA HOSP, DEPT PHYSIOL, S-10401 STOCKHOLM 60, SWEDEN (Reprint); KAROLINSKA INST, KAROLINSKA HOSP, INST ENVIRONM MED, S-10401 STOCKHOLM 60, SWEDEN; KAROLINSKA INST, KAROLINSKA HOSP, DEPT UROL, S-10401 STOCKHOLM 60, SWEDEN
CYA SWEDEN
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1991) Vol. 141, No. 1, pp. 49-56.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 36
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Intravital microscopy of rabbit tenuissimus muscle was used for studies of endogeneous nitric oxide as a microvascular regulator in vivo. Derivatives of **arginine** were **administered** in order to modulate the formation of nitric oxide from L-arginine. N-omega-nitro-L-arginine methylester (L-NAME) (1-100 mg kg-1 i.v.) dose-dependently reduced microvascular diameters. A concomitant blood pressure increase and a decrease in heart rate was observed. The blood pressure increase induced by L-NAME (30 mg kg-1) was reversed by L-arginine (1 g kg-1) but not D-arginine. Vasodilation in response to topical acetylcholine (0.03-3-mu-M) was significantly inhibited by L-NAME (30 mg kg-1), whereas vasodilation by sodium nitroprusside (300 nM) was not affected. Vasomotor nerve-induced vasodilatation, induced by stimulation of the tenuissimus nerve after neuromuscular blockade by pancuronium in animals pretreated with guanethidine, was significantly attenuated by L-NAME, an effect also reversed by L-arginine. The vasodilatation in response to active contractions of the muscle induced by motor nerve stimulation as well as the vasodilator response elicited by graded perfusion pressure reductions were unaffected by L-NAME or N(G)-monomethyl-L-**arginine** (L-NMMA, 10(-4) M) **administered** topically.

Our results indicate that endogenous nitric oxide formed from L-arginine is a modulator of microvascular tone in vivo. Furthermore, the results suggest that endogeneous nitric oxide is required for vasomotor nerve-induced vasodilatation, whereas it does not appear to play a role in myogenic vasodilatation or functional hyperaemia in this tissue.

L12 ANSWER 1988 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:52162 SCISEARCH
GA The Genuine Article (R) Number: ET375
TI ROLE OF CENTRAL MINERALOCORTICOID BINDING-SITES IN DEVELOPMENT OF HYPERTENSION
AU JANIAK P C; LEWIS S J; BRODY M J (Reprint)
CS UNIV IOWA, COLL MED, DEPT PHARMACOL, BOWEN SCI BLDG, IOWA CITY, IA, 52242
CYA USA
SO AMERICAN JOURNAL OF PHYSIOLOGY, (1990) Vol. 259, No. 5, pp. R1025-R1034.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The possibility that central mineralocorticoid binding sites are involved in the development of mineralocorticoid hypertension was examined using chronic blockade of these sites with a specific mineralocorticoid

receptor antagonist RU 28318 administered by intracerebroventricular (icv) infusion. The antagonist significantly attenuated the development of deoxycorticosterone acetate (DOCA)-salt hypertension, but the development of one-kidney, one-clip renal hypertension was not affected. This antihypertensive action was attributable to a central action, since intraperitoneal infusion of the same dose of mineralocorticoid antagonist did not alter the peak development of DOCA-salt hypertension. The icv infusion of RU 28318 did not change either the increase of fluid intake induced by DOCA-salt treatment or the pressor reactivity to centrally or peripherally injected **arginine** vasopressin and angiotension II and peripherally **administered** phenylephrine. The antihypertensive action of icv infusion of the mineralocorticoid antagonist was associated with a reduction of neurogenic vasomotor tone and a restoration of impaired arterial baroreflexes. We conclude that functional integrity of central mineralocorticoid binding sites is required for the full development of DOCA-salt hypertension.

L12 ANSWER 1989 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:17827 SCISEARCH
GA The Genuine Article (R) Number: EQ098
TI EFFECTS OF PERIPHERALLY **ADMINISTERED ARGININE** -VASOPRESSIN ON LEARNING, RETENTION AND FORGETTING IN MICE
AU ALESCIOLAUTIER B (Reprint); SOUMIREUMOURAT B
CS UNIV PROVENCE, IBHOP, CNRS, URA 372, NEUROBIOL COMPORTEMENTS LAB, TRAVERSE CHARLES SUSINI, F-13388 MARSEILLE 13, FRANCE (Reprint)
CYA FRANCE
SO BEHAVIOURAL BRAIN RESEARCH, (1990) Vol. 41, No. 2, pp. 117-128.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 38
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The effects of peripheral injections of (Arg)-vasopressin were investigated on different stages of memory processes using an appetitive visual discrimination task and a one-trial passive avoidance conditioning in mice. The peptide was administered at one of two doses: 50-mu-g/kg or 25-mu-g/kg. The main effects of vasopressin were observed only for the higher dose. Concerning pre-session vasopressin administration in the visual discrimination task, the effect of the peptide seemed to depend on the level of learning reached at the time of treatment. Indeed, we observed a deleterious effect of vasopressin on learning capacities when the peptide was administered before the first learning session, a bimodal effect (either an improvement or an impairment) on performance when the peptide was administered before the second learning session and an important enhancement of retention performance when the peptide was administered before the retention session, performed 24 days after training. When postsession vasopressin administration was assessed, an improvement of performance was observed indicating a facilitatory effect of vasopressin on consolidation processes. When passive avoidance conditioning was used, an enhancement of retention performance was registered only when the peptide was injected before the retention session at the 50-mu-g/kg dose. No facilitation was observed for the 25-mu-g/kg dose whatever the experimental condition was, i.e. post-learning or pre-retention injection. In order to test eventual non-specific effects of vasopressin, the influence of the peptide on locomotor activity was assessed before the two doses. The results show an important reduction of locomotor activity with the 50-mu-g/kg dose, during 4 h following vasopressin injection. No effect was observed with the 25-mu-g/kg dose. The whole results suggest that vasopressin-induced hypoactivity can directly influence the subsequent learning performance when the treatment was performed in pre-session situations. However, when the level of information is sufficient and beyond the direct effect of the drug, a memory effect may be considered with the 50-mu-g/kg dose independently from the locomotor effect, when the treatment was delivered during

consolidation period (post-session) or in long-term retrieval situation (pre-session).

L12 ANSWER 1990 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 89:366400 SCISEARCH
GA The Genuine Article (R) Number: AE622
TI DISTRIBUTION AND METABOLIC-FATE OF RADIOACTIVE CARBON FROM L-[U-C-14] **ARGININE ADMINISTERED** INTO MICE
AU GOTO H (Reprint)
CS OSAKA MED COLL, DEPT ANAT, TAKATSUKI, OSAKA 569, JAPAN (Reprint)
CYA JAPAN
SO ACTA HISTOCHEMICA ET CYTOCHEMICA, (1989) Vol. 22, No. 2, pp. 215-225.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 27

proconvulsant effects probably mediated by an increase in NO synthesis.

L12 ANSWER 1985 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:253480 SCISEARCH
GA The Genuine Article (R) Number: FH481
TI STUDIES ON THE PRECURSOR OF METHYLGUANIDINE IN RATS WITH RENAL-FAILURE
AU YOKOZAWA T (Reprint); FUJITSUKA N; OURA H
CS TOYAMA MED & PHARMACEUT UNIV, DEPT APPL BIOCHEM, WAKAN YAKU RES INST, SUGITANI, TOYAMA 93001, JAPAN (Reprint)
CYA JAPAN
SO NEPHRON, (1991) Vol. 58, No. 1, pp. 90-94.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 27
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Each of creatinine (Cr), guanidinoacetic acid (GAA) and **arginine** (Arg) was **administered** intraperitoneally to rats with renal failure, and the levels of methylguanidine (MG) in the serum, liver, kidney, muscle and urine were determined at certain intervals. The levels of MG in the serum, liver, kidney, muscle and urine after Cr administration increased markedly with time. The amount of total MG at 24 h was estimated to be 114-mu-g/100 g body weight, which accounted for 0.46% of the Cr dose. In contrast, changes in MG levels after administration of GAA or Arg were only slight in comparison with those after Cr administration. Thus, MG was proved to be produced mainly from Cr.

L12 ANSWER 1986 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 91:176046 SCISEARCH
GA The Genuine Article (R) Number: FC708
TI EFFECT OF EXOGENOUS ARGININE VASOPRESSIN ON ADRENOCORTICOTROPIN AND CORTISOL RELEASE IN MYOTONIC-DYSTROPHY PATIENTS - DELAYED-RESPONSES OF NORMAL MAGNITUDE
AU GRICE J E; JACKSON J; HEWETT M; PENFOLD P J; JACKSON R V (Reprint)
CS UNIV QUEENSLAND, GREENSLOPES HOSP, DEPT MED, NEUROENDOCRINE RES UNIT, BRISBANE, QLD 4120, AUSTRALIA
CYA AUSTRALIA
SO JOURNAL OF NEUROENDOCRINOLOGY, (1991) Vol. 3, No. 1, pp. 65-68.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 24
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We **administered** intramuscular **arginine** vasopressin (AVP) to ten normal controls and eight myotonic dystrophy patients. By measuring plasma AVP levels in five of the myotonics and all the normals, we showed that absorption and distribution of AVP was not delayed or significantly reduced in myotonics. The magnitude of the mean plasma adrenocorticotropin (ACTH) response to AVP in the myotonics was not different from that of normals, although it was significantly delayed (mean peak time, 37.5 +/- 4.9 versus 17.0 +/- 3.2 min). We propose that this delay was caused by a significantly reduced ACTH secretion rate in myotonics, because the maximum rate of detection of ACTH in plasma is reduced in myotonics (0.6 +/- 0.2 versus 1.7 +/- 0.5 pmol/L/min), whose corticotropes, while having the same capacity to respond to the AVP stimulus, are slower to attain that capacity. The mean integrated cortisol response (AUC) was significantly smaller for myotonics (8072 +/- 2017 versus 13049 +/- 1630 nmol.min/L). This may be due to the slower rate of ACTH delivery to the adrenal in myotonics. The timing of the adrenal response does not appear to be impaired in myotonic dystrophy, with the cortisol peak following the ACTH peak by approximately 15 min in both groups. The normal magnitude ACTH response to AVP in myotonics is in contrast to that seen to ACTH secretagogues acting via

=> s administer####(6a)arginin###
L12 2006 ADMINISTER####(6A) ARGININ###

=> d l12 1975-1990 bib,ab

L12 ANSWER 1975 OF 2006 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 92:33069 SCISEARCH
GA The Genuine Article (R) Number: GX687
TI EFFECT OF AN ARGININE ANALOG ON ACETYLCHOLINE-INDUCED CORONARY MICROVASCULAR DILATATION IN DOGS
AU KOMARU T; LAMPING K G; EASTHAM C L; HARRISON D G; MARCUS M L; DELLSPERGER K C (Reprint)
CS UNIV IOWA, COLL MED, DEPT INTERNAL MED, E329-1GH, IOWA CITY, IA, 52242; UNIV IOWA, COLL MED, CTR CARDIOVASC, IOWA CITY, IA, 52242
CYA USA
SO AMERICAN JOURNAL OF PHYSIOLOGY, (DEC 1991) Vol. 261, No. 6, Part 2, pp. H2001-H2007.
ISSN: 0002-9513.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 40
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The purpose of this study was to elucidate the contribution of endothelium-derived relaxing factor (EDRF) derived from arginine to acetylcholine (ACh)-induced coronary arteriolar vasodilatation in vivo. Experiments were performed in 62 open-chest anesthetized dogs. Internal diameters of small arterioles (< 120-mu-m) and large arterioles (> 120-mu-m) were measured using an intravital microscope and stroboscopic epiillumination synchronized to the cardiac cycle. Topically **administered** N(G)-monomethyl-L-**arginine** (L-NMMA, 3 x 10(-4) M) constricted small arterioles (-10.7 +/- 3.1% from control diameter, P < 0.05), but L-NMMA did not produce vasoconstriction in large arterioles. ACh, in the absence of L-NMMA, caused a dose-dependent vasodilatation in both small and large arterioles. In large arterioles, L-NMMA completely abolished the ACh-induced vasodilatation (10(-5) M topical ACh: from 13.3 +/- 3.0 to -2.0 +/- 1.5%, P < 0.05; 10(-4) M ACh: from 20.9 +/- 3.9 to -3.0 +/- 1.9%, P < 0.01). In small arterioles, L-NMMA only partially inhibited the vasodilatation (10(-5) M ACh: from 35.4 +/- 4.0 to 19.0 +/- 2.7%, P < 0.05; 10(-4) M ACh: from 42.5 +/- 4.8 to 22.6 +/- 3.1%, P < 0.05). L-Arginine (10(-3) M topically) reversed L-NMMA inhibition of ACh-induced vasodilatation. Persistent dilatation of small arterioles also occurred when N(G)-nitro-L-**arginine** rather than L-NMMA was **administered**. Neither K+ channel blockers [glibenclamide (10(-5) M) and tetraethylammonium (3 x 10(-2) M)] nor indomethacin (5 x 10(-5) M) had an additional inhibitory effect on ACh-induced vasodilatation in the presence of L-arginine analogue. These data suggest that EDRF derived from arginine modulates basal tone in small arterioles but that arginine is unlikely to be the only source of factors that modulate ACh-induced vasodilatation in these vessels. In contrast, a nitrosyl compound derived from arginine exclusively accounts for ACh-induced vasodilatation in large arterioles.

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AN 92:3419 SCISEARCH
GA The Genuine Article (R) Number: GV707
TI INHIBITION OF UREA TUBULAR REABSORPTION BY PGE1 INFUSION IN MAN
AU CONTE G (Reprint); CIANCIARUSO B; DENICOLA L; SEPE V; ROMANO G; DOMENICO R; CAGLIOTI A; FUIANO G; DALCANTON A
CS NAPLES UNIV, FAC MED 1, DEPT NEPHROL, VIA LUIGI CALDIERE 10, I-80128 NAPLES, ITALY (Reprint); NAPLES UNIV, FAC MED 2, DEPT NEPHROL, I-80128 NAPLES, ITALY; UNIV REGGIO CALABRIA, FAC MED CATANZARO, CALABRIA, ITALY
CYA ITALY